Mental and multiple disabilities

Concept of a therapy system

Remarkable progress has been made in the last 30 years by medical therapy for mentally handicapped – in comparison with a stagnation that had lasted for centuries. The impulses came from various marginal areas of medicine and pedagogy but to a lesser extent from principal areas such as neurology and psychiatry. Physiology, biochemistry and - morphology created a better understanding of the structure and functions of the nervous system; pediatricians, physiotherapists and pedagogues worked out practical techniques for treatment.

Let us again summarize the contrast between present and past. In the past, people were helpless when faced with the manifestations of mentally handicapped persons or diseases of the central nervous system. This helplessness was reflected by isolating and tranquilising the patient; that is, the central nervous system was not trained and depressed. But in our days the active principle of encouragement and training has basically found general acceptance though not everywhere in practice. In terms of historical development, we should have overcome the periods of eliminating the handicapped (in antiquity and remnants of that age in modern times), of isolating the patient (behind big walls in medieval times). The main duty of medicine in our times is the integration of handicapped people into the environment. Despite basic progress and positive trends, individual cases still nowadays point out the limitations of these possibilities – but also the limitations of quite a few erroneous developments in terms of organization. Long years of my own experience on more than 5000 cases of handicapped children have shown that the potential offered by therapy is in most instances utilized only in a unilateral and thus incomplete manner; barriers erected in terms of organization often obstruct rather than favour the development of these children. A few medical and educational examples are intended to clarify this statement.

The Drawbacks of Specialization

Certainly, *medical gymnastics, physiotherapy, stimulation-programms* have been and still are one of the most important elements of therapeutic progress. In the course of the change of designation from «infantile cerebral paresis» to «cerebral motor disturbance» remedial gymnastics – primarily through specialized methods breathing sectarian intolerance – are emphasized to such an extent – both in the projecting line of thought and in terms of time – that other methods of treatment are neglected or can no longer be accommodated in terms of time.

The *introduction of anticonvulsive agents* was a step forward which took the notion of unavoidable fate from the seizure conditions. Wherever such progress became an end in itself, where the symptom of the «convulsive cerebral attack» was made the centre of the condition and the sole target on which therapy should focused, the usefulness of symptomatical treatment may be perverted to damage to the personality of the patient; this may happen due to a neglect of other ways of treatment and the suppression of the central nerve functions. The gamut of the disadvantages of a one-sided therapy ranges from serious damage to the ske-

leton (Rachitis anticonvulsiva) to «Morbus anticonvulsivus» where the consequences of a high-dosage and unsuitable anticonvulsive medication are of a more serious pathological nature than the seizure condition proper.

The system of our *specialized pedagogy* may be compared with an excellent network of roads where, unfortunately, there are no cross-connections. The patient who, due to geographical location, is put on such a road (i.e. a special school G for Mentally handicapped, special school L for Learning handicapped, school for physically handicapped), will as a rule have to keep on that road since he is given the result of an intelligence test as a starting position to serve as an identification card.

The *field of testing* (psychological, behavioural, intelligence tests and others) is highly diversified with its more than 1000 test methods. However, practical experience shows that the result often testifies more to the abilities of the tester than to the abilities of the tested patients. A handicapped child who lives in contact with one or a few reference persons in his environment, will never show the same performance which he is able to show when confronted with strangers; often any cooperation is refused during the test. An intelligence quotient (IQ) labelled from such test situations is an unsurmountable obstacle for many children who are to be given adequate supportin their development.

Handicap as an Integral Concept

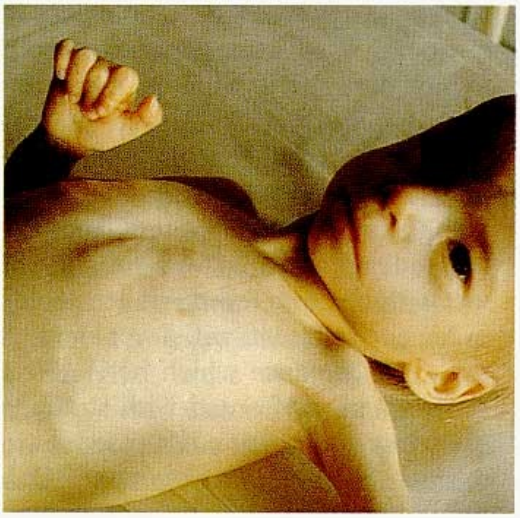
The development of the child, that is the evolution of the biological potentials contained in the genotype in accordance with the laws of nature, is a complex process composed of a somatic and an intellectual (mental) group of factors; the physical area includes anthropometric data (length of body, weight, circumference of head, growth), and statomotoric functions. The mental area consists of psychic, social and intellectual components. In handicapped patients the individual components are usually not affected in uniform manner; the assessment (often identical with diagnosis) is formed in most instances, according to the most serious deficiency (spastics, debility, numbness, speech disorder, ataxia). Here it is often overlooked that the patient is a personality who, besides his shortcomings, has also positive (and in this case often above-average) qualities (for example social attitudes, willingness to give help, tidiness, a good instinct).

An optimized therapeutic concept must be based on the integrality of the patient who is handicapped, primarily recognize the mosaic of symptoms, evaluate it and work out an individual scheme for treatment and guidance from the individual possibilities and shortcomings. The supreme therapeutic target must be the encouragement of personal development, not the elimination of a symptom.

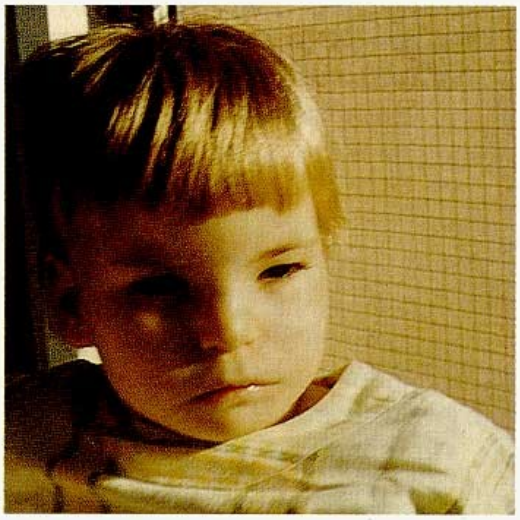
Diagnostic Requirements

Looking at the changes that have occurred in the judgment and classification of mental handicaps in the last 50 years by referring to the diagnoses used, which ranged from

«Vitium cerebri»
over
«Oligophrenia» (debility, imbecility, idiocy)
to



a



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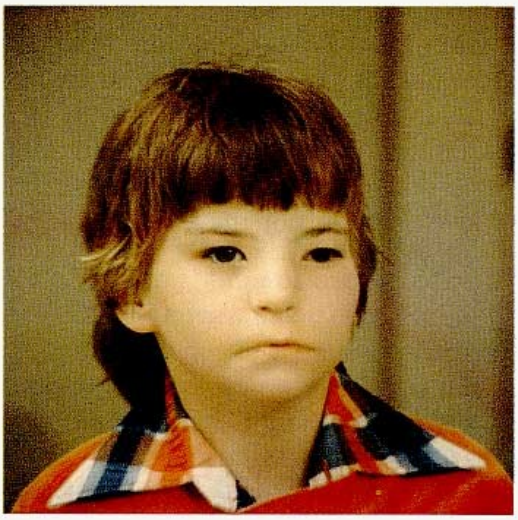

c

«Cerebral palsy»
(infantile cerebral paresis),
«cerebral motoric disorders»
and finally to
«brain damage in early childhood»,

two things are evident:

As against the general organ-related diagnosis, individual symptoms (such as the degree of intelligence, motoric handicap, seizure conditions) were given predominant attention in the course of time and promoted to « Diagnosis» status. The provisional end of this trend, the «brain damage of early childhood» is to be assigned to the period where, about 150 years ago, developments started:

Fig. 258:
Progeria series of development 710812-780523

a)
The boy of $3\frac{5}{12}$ years was admitted with 75 cm stature (-24 cm $= -25\%$), 5.56 kg bodyweight (-4.5 kg related to stature = about -75%(!) -9 kg corresponding to age = about -150%) because he was unable to ingest food. Dystrophic, senile appearance, frontal vessels as thick as pencils. No reaction to optic, acoustic stimuli, no sounds, little spontaneous movement. On polyvitamins, digestive ferments, primobolan gains in weight up to 12.9 kg, increase of stature to 80 cm in 7 months, but no static and mental progress.
At $4\frac{3}{12}$ years, implantation of 100 mg of cerebral lyophilisate. Slowly beginning, then rapidly visible motor and mental development; at $5\frac{4}{12}$ years 92 cm (-19 cm), 17.2 kg, according to age.

b)
At $5\frac{8}{12}$ years 98 cm (-16 cm), 18.4 kg. Implantation: 100 mg diencephalon, 100 mg frontal brain; grows 3 cm in 40 days; reacts to environment, sits, stands, walks, utters first sounds.

c)
At $10\frac{6}{12}$ years, the boy attends special school, speaks indistinctly owing to Moebius's syndrome, fully integrated in social respect, 126 cm (-12 cm), 30.5 kg. At 12 years, 4th class of special school, reads, writes, counts up to 20, is interested in everything; 133 cm, 31.8 kg.

Tab. 32: **Important diagnostic partial symptoms of mental retardation and multiple physical disability**

somatic		mental		Sens-organs	Peculinities
anthropo-metric	stato-motoric	psycho-social	intellectual		
general growth anomalies nanism microsomia high stature giantism dystrophy adiposis **partial growth anomalies** microcephalia macrocephalia dyscephalia hypognathia hypergnatia hypogeny hypergeny brachymelia brachycarpia brachydactylia brachy-mesophalangy acromicria dolichomelia dolichocarpia dolichodactyly acromegalia cranial hypoplasia (bird's head) caudal hypoplasia dysraphia dysmelia	**muscular hypertony** monoplegia diplegia triplegia tetraplegia (Little-S.) hemiplegia **muscular hypotonie** Foerster-S. puppet phenomenon «Floppy infant» **muscular dystony** **Dyskynesia** athetosis chorea chorea-athetosis **ataxia**	apathie hyperkinesis erethism aggressivity listlessness helplessness tyrant uncleanliness autism	debility imbecillity idiocy partially disturbed performance no abstract thinking no discrimination absent-mindedness echolalia perseveration cortical blindness cortical deafness loss of initiative	**skin** analgesia hypalgesia trophic disturbances disturbed circulation anomalous pigmentation **speech** lacking understanding of speech motoric speech disturbance (dyslalia) **hearing** hardness of hearing deficient selective pitch of voice deafness **vision** **strabism** nystagmus microphthalmy macrophthalmy opacity of lense retrolentary fibroplasia glaucoma amblyopia blindness	increased predisposition to infections lacking immunoglobulins **gastro-intestinal disorders** inappetence lack of ferments chronic obstipation chron. gastritis ulcera oesophagitis gastro-oesophageal reflux frequent vomiting **vegetative symptoms** vegetative lability disturbed peripheral circulation acrocyanosis trophic disorders lost or weakened regulation of body-temperature

There is merely a difference in degree compared with «Vitium cerebri», no basic difference. But the most dangerous attribute is the Intelligence Quotient (IQ), a figure computed with sources of error; such figure lends itself to easy processing by government offices but often entails disastrous consequences for the treatment and encouragement of the children because, in many instances, the qualification for being encouraged, the possibility of treatment and the type of schools to be attended are determined with this figure.

Starting with this negative aspect it is pointed out that the mosaic of a handicap is composed of individual symptoms which should also be covered and formulated in each single instance; otherwise we lose sight of them in our therapy. There is a momentous difference as to whether the label of a handicap reads:

a) brain damage in early childhood or
b) motoric and mental retardation;
   athetosis;
   ataxia;
   strabism;
   dyslalia.

Tab. 33: **Multifactorial damages**

anencephaly	arthromyodysplasia (arthrogryposis)
hydrancephaly	Hallervorden-Spatz degeneration
arhinencephaly	Sturge-Weber's syndrome
parencephaly	(trigeminus-angiomatosis)
microcephaly	syndrome with aniridia, cerebellar
hydrocephaly	ataxia
clefts of lips, maxilla, palate	oligophrenia
rachischisis	Pierre Robin's dysmorphia
lacunar skull	bird-face (Seckel's nanism)
dysmorphia-syndromes	Hallermann Streiff-disease
Cornelia de Lange-syndrome	(oculomandibulo-dyscephalia with
Rubinstein's syndrome	hypotrichosis)
	Many more, mostly rare, syndromes

Tab. 34: **Environmental Factors which may lead to mental damage or impairment:**

Prenatal Damage	**Natal Dangers**
Maternal Noxae:	Immaturity
Nicotine abuse	Prematurity
Alcoholic abuse	Umbilical-cord strangulation
Drug abuse	Prolonged birth
Antiepileptic therapy	Difficult birth
Cytostatica	Prolonged hypoxia
Radiation damage	Anoxia
Infections:	Artificial aids (forceps, suction cup)
Bacterial sepsis	
E. Coli	**Postnatal Damage**
Pyoceanus	Encephalo-enteritis (toxicosis)
Proteus	Encephalitis
Staphylococcos	Vaccination encephalopathy
Beta-hämol. B-Streptococcos	Meningitis
German measles	Subdural hematome-hygroma
Virus conditions	Anaesthesia accidents
Toxoplasmosis	Cerebral angiographies
Cytomegalia	Traumatic damage of central nerve system
Lues	cardiac arrest
Anemia	Chronic hypoxia
Bleedings (hemorrhages)	Cerebral seizures
Toxicosis	Athyreosis-hypothyreosis
Nidation anomalies of the fetus	Hypocalcemia – hypercalcemia
Blood group incompatibility	Hypoglycemia

Under a) there is a statement not related to any target, it does not lead to a therapeutic consequence;

Under b) there is a comprehensive stock-taking with direct therapeutic demands (see Table 32):

The diagnosis that serves as a premise for a therapy suitable for the individual case, should include the following basic elements:

Fig. 259:
Alcoholic-embryo-fetopathy: retarded development, retarded development of speech, mimic expression (a) before and (b) after two treatments with implantations 2 years later; most distinct is the progress in speech development.

Tab. 35: **Infantile cerebral paresis**

Hypertonic Forms Muscle hypertonia Spastic monoplegia diplegia triplegia tetraplegia Spastic hemiplegia	**Dyskinetic Forms** Chorea (motoric restlessness) Athetosis (motoric stiffness) Choreo athetosis (coexistence and change of motoric restlessness and spasm) Fine motor «clumsiness»
Hypotonic Forms Muscle hypotonia Stuffed doll syndrome «Floppy infant»	**Atactic Forms** Cerebellar ataxia Cerebello-spinal ataxia
Dystonic Forms Changes of hypertonia and hypotonia	**Mixed Forms** Combinations of the above listed symptoms among each other with sensory deficiencies, trophic disturbances, reduced intelligence and psychic deviations.

1. Anthropometric data (length of body, body weight, proportions);
2. Size of skull and shape of skull
   a) visual judgment;
   b) circumference of skull (= surface measurement of skull basis);
   c) Radiological skull measurements including the volume index of the skull;
3. Analysis of development (fig. 263)
   a) stato-motoric;
   b) fine-motoric, coordination;
   c) eating, drinking, speaking;
   d) social development, psychic development;
   e) intellectual performance;
4. Neurological symptoms (fig. 264).
5. Anthropological age (bone age).

6. Electroencephalogram (EEG). Additional data can be obtained for special areas, which may be relevant for therapy:
7. Echoencephalogram;
8. Computer Tomogram;
9. Audiometry;
10. Psychological tests;
11. Eye examinations;
12. Analyses of the metabolism;
13. Diaphanoscopy;
14. Cerebral angiography.

Genesis and Types

Most classification principles concerning handicapped conditions are based on the genesis and the clinical symptoms or represent a complex of criteria.

We can distinguish the following main groups based on the genesis:

1. *Hereditary metabolic disorders (Tab. 31);*
2. *Chromosomal aberrations (Tab. 23);*
3. *Environmental handicaps (Tab. 34);*
4. *Multifactorial damages (Tab. 33).*
5. *Types of infantile cerebral paresis (Tab. 35)*
6. *Partial disturbances of performance.*

The summaries in Tab. 29–35 and in the following list give a concise survey of the total field.

Partial disturbances of performance

The probably mildest forms of consequences of brain damage in early childhood are the so-called partial disturbances of performance in various areas of the motoric system, particularly of the learning process. Such partial disturbances may be located within the mosaic of infantile cerebral palsy, but also occur as individual symptoms. The following forms must be distinguished:

1. *Disorders of reading techniques*

a) Incorrect vowels (for example I instead of A);
b) Incorrect consonants;
c) Reversals (e.g. pit/tip);
d) Omission of sounds or added sounds (for example ... un instead of sun; shipi instead of ship);
e) Substitutions (instead of saying «I live in Aschaffenburg»/«my home is in Aschaffenburg»);
f) Repetitions (for example the ca, ca, cat or the cat, cat, cat);
g) New words added or omitted (e.g. instead of saying «a dog» the child says «a vicious dog»; ...was a king instead of there was a king);
h) Refusals (e.g. the sentence «one of the most wonderful experiences ...» is spoken as follows: one of the experiences ...
i) Inability to articulate unknown words, while the other phonetic structures are degenerate;
j) Deficient differentiation of letters, parts of words and syllables (e.g. between bead and bed);
k) Lack of ability to discover differences between sounds and words;
l) Difficulty in observing lines;
m) Difficulty in proceeding from the right side to the next line below on the left;
n) Poor understanding of the material read;

2. *Writing-disorders*

a) Delayed and slow learning to write;
b) Writing in reverse;
c) Letter size cannot be coordinated;
d) Slipping characters;
e) Strikingly irregular line thickness;

f) Legasthenia;

3. Speech disorders

a) Disorders when spelling (e. g. loss of letters, telegraph style words);
b) Inability to spell, that is to differentiate the individual letters contained in a word;
c) Poor understanding of language;
d) Disturbances of language motor system (difficulty in pronouncing);
e) Difficulty in finding words;
f) Poor structure of sentences (difficulty in establishing the right word order);
g) Difficulties in orientation;
h) Stuttering and stammering.

Possibilities and limitations in biological development

The central nervous system of man is the sole organ system the «fetal stage» of which is not completed at the time of birth. Processes of fetal maturation and differentiation extend up to the 4th year of life; they are completed only upon maturation of the medullary sheath. This results in a few essential aspects for vulnerability and therapeutic possibilities and limitations. The following facts should be kept in mind (fig. 260):

1. With a maturely born child the final number of nerve cells is already present; no new ones will be added during his lifetime.
2. Despite the final cell number the weight of the brain of a mature newborn is 350 g on the average; at the end of the somatic growth it is 1250 g on an average, the number of cells being the same.
3. Without increasing the number of neurons the brain volume triples $3\frac{1}{2}$ times by way of formation of secondary structures of the neuron.
4. The main increase in brain volume (= weight) takes place in the first three years of life, that is in the «caught-up» fetal period of the central nervous system. About $\frac{3}{4}$ of the

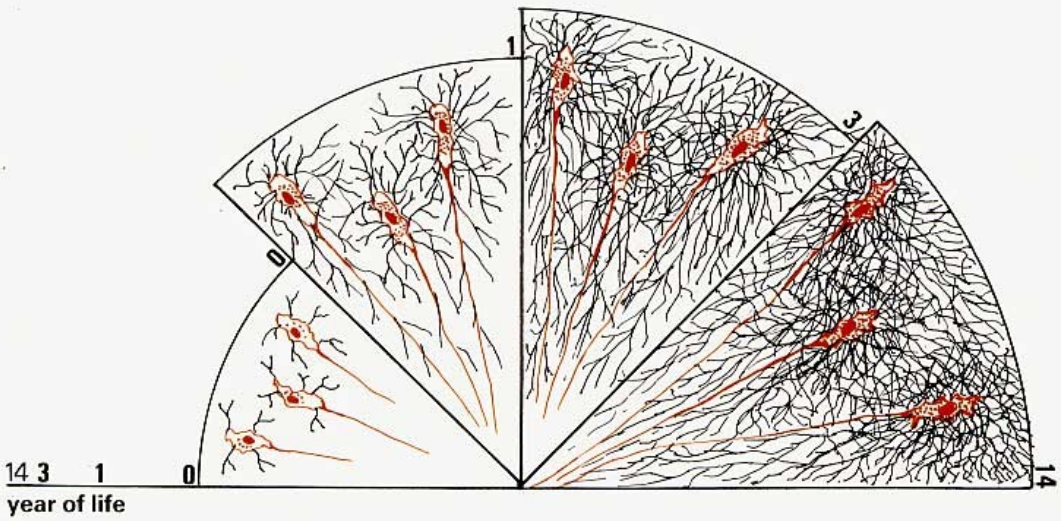

Fig. 260: Maturation of the brain from birth to the 14th year of life. By way of formation of secondary structures the brain volume (= weight) increases $3\frac{1}{2}$ fold; the number of neurons present at the time of birth is not changed.

postnatal brain growth takes place in this age period.

These biological laws result in a higher vulnerability of the central nervous system in the first four years of life but also the possibility of acting on it by way of therapy. In particular, the first three years offer chances and potentials for therapy applications with biological «building stones» and a training program from the periphery. These possibilities are no longer available after the fourth year. Possibilities of treatment not utilized in the first three years of life will remain wasted chances throughout life. Even with an increased therapeutic input and effort they can be corrected only in part.